Acute Compartment Syndrome

49

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Key Points

- Compartment syndrome is defined as an elevation of the interstitial pressure in a closed osseo-fascial compartment that results in microvascular compromise.
- High suspicion is mandatory when risk factors are present: missing the diagnosis could be lethal.
- In the case of abdominal compartment syndrome, bladder pressure measurement is considered the diagnostic gold standard.
- In the case of acute compartment syndrome of extremities, clinical exam is sufficient to make the diagnosis.
- Surgical decompression is the main treatment for acute compartment syndrome (both abdominal and of the extremities).
- Compartment syndrome could happen in every compartment of the body.

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49.1 Introduction

The first report of a syndrome that could be called "compartmental" is by Dr. Richard von Volkmann in 1881 [1]. The Volkmann's syndrome occurred after the application of splints or bandages to the upper extremity and was characterized by an initial paralysis and a subsequent flexion contracture of the hand and forearm. Volkmann believed the syndrome was the result of an ischemic process due to impaired arterial perfusion leading to muscle cell death.

In the early twentieth century, Jepsen reproduced the observations by Volkmann on animal models: he showed that limb fractures acted like splints or bandages rising compartment (defined as a closed space surrounded by muscular aponeurosis) pressure. When the compartment pressure reached the perfusion pressure, blood flow stopped, tissue death occurred, and the syndrome appeared [2].

With time, authors described many syndromes sharing the pathogenesis reported by Jepsen, and nowadays, thanks to Matsen [3], any disorder characterized by "an elevation of the interstitial pressure in a closed osseo-fascial compartment that results in microvascular compromise" is classified as compartmental.

In the present chapter, we aim to face syndromes that may be faced by emergency surgeons: the abdominal compartment syndrome and the acute compartment syndrome of the limbs.

49.2 Abdominal Compartment Syndrome

49.2.1 Definition

The abdominal compartment syndrome is defined according to the Cartagena 2013 WCACS (World Congress of the Abdominal Compartment Society) [4] reported in Table 49.1. Specifically, ACS (abdominal compartment

P. Aseni et al. (eds.), *Operative Techniques and Recent Advances in Acute Care and Emergency Surgery*,
https://doi.org/10.1007/978-3-319-95114-0_49

Table 49.1 Abdominal compartment syndrome definitions according to the Cartagena WCACS 2013

Definition 1	IAP is the steady-state pressure concealed within the abdominal cavity
Definition 2	The reference standard for intermittent IAP measurements is via the bladder with a maximal instillation volume of 25 mL of sterile saline
Definition 3	IAP should be expressed in mmHg and measured at endexpiration in the supine position after ensuring that abdominal muscle contractions are absent and with the transducer zeroed at the level of the midaxillary line
Definition 4	IAP is approximately 5–7 mmHg in critically ill adults
Definition 5	IAH is defined by a sustained or repeated pathological elevation in IAP C 12 mmHg
Definition 6	ACS is defined as a sustained IAP > 20 mmHg (with or without an APP < 60 mmHg) that is associated with new organ dysfunction/failure
Definition 7	IAH is graded as follows: Grade I IAP 12–15 mmHg; Grade II IAP 16–20 mmHg; Grade III IAP 21–25 mmHg; Grade IV IAP > 25 mmHg
Definition 8	Primary IAH or ACS is a condition associated with injury or disease in the abdominopelvic region that frequently requires early surgical or interventional radiological intervention
Definition 9	Secondary IAH or ACS refers to conditions that do not originate from the abdominopelvic region
Definition 10	Recurrent IAH or ACS refers to the condition in which IAH or ACS redevelops following previous surgical or medical treatment of primary or secondary IAH or ACS
Definition 11	APP = MAP − IAP
Definition 12	A polycompartment syndrome is a condition where two or more anatomical compartments have elevated compartmental pressures
Definition 13	Abdominal compliance is a measure of the ease of abdominal expansion, which is determined by the elasticity of the abdominal wall and diaphragm. It should be expressed as the change in intra-abdominal volume per change in IAP
Definition 14	The open abdomen is one that requires a temporary abdominal closure due to the skin and fascia not being closed after laparotomy
Definition 15	Lateralization of the abdominal wall is the phenomenon where the musculature and fascia of the abdominal wall, most exemplified by the rectus abdominus muscles and their enveloping fascia, move laterally away from the midline with time

ACS abdominal compartment syndrome, *APP* abdominal perfusion pressure, *IAH* intra-abdominal hypertension, *IAP* intra-abdominal pressure, *MAP* mean arterial pressure

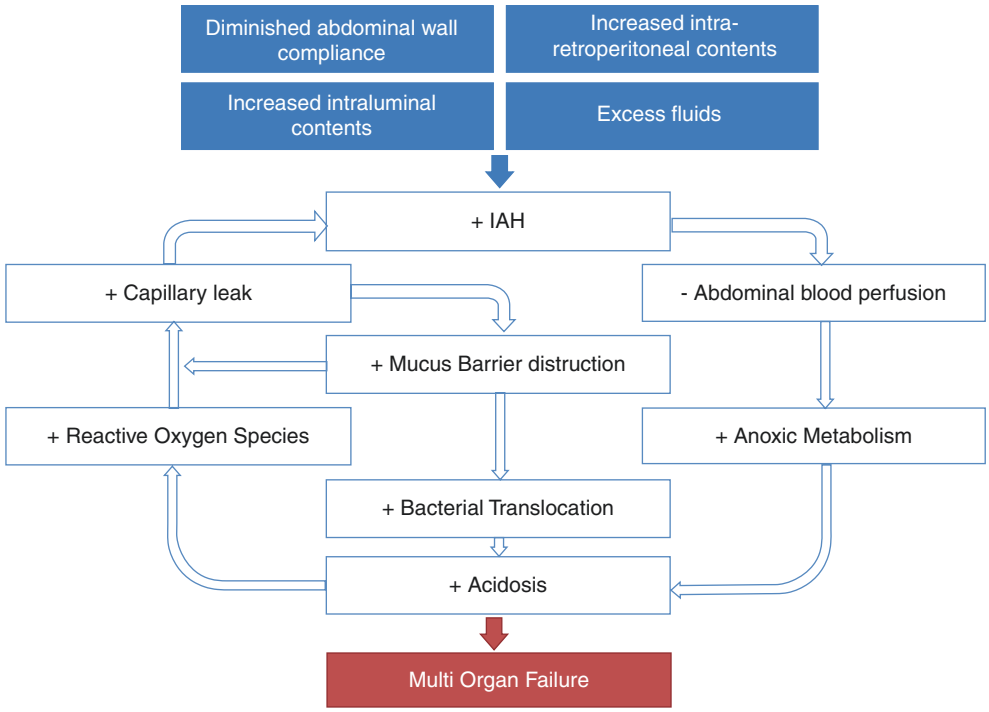

Fig. 49.1 Pathophysiology of the abdominal compartment syndrome

syndrome) is defined as a sustained IAP (intra-abdominal pressure) >20 mmHg (with or without an abdominal perfusion pressure (APP) <60 mmHg) that is associated with new organ dysfunction/failure. Figure 49.1 summarizes the pathophysiology of the abdominal compartment syndrome.

49.2.2 Common Etiology and Risk Factors

On a general basis, IAH (intra-abdominal hypertension) and ACS develop if one of four main conditions occurs abruptly: (1) diminished abdominal wall compliance, (2) increased intraluminal contents, (3) increased intra-retroperitoneal

Table 49.2 Abdominal compartment syndrome risk factors according to the Cartagena WCACS 2013

Diminished abdominal wall compliance	*Increased intra-luminal contents*	*Others/miscellaneous*
Abdominal surgery	Gastroparesis/gastric distention/ileus	Age
Major trauma	Ileus	Bacteremia
Major burns	Colonic pseudo-obstruction	Coagulopathy
Prone positioning	Volvulus	Increased head of bed angle
		Massive incisional hernia repair
Increased intra-abdominal contents	*Capillary leak/fluid resuscitation*	Mechanical ventilation
Acute pancreatitis	Acidosis	Obesity or increased body mass index
Distended abdomen	Damage control laparotomy	PEEP
Hemoperitoneum/pneumoperitoneum or intra-peritoneal fluid collections	Hypothermia	Peritonitis
Intra-abdominal infection/abscess	Increased APACHE-II or SOFA score	Pneumonia
Intra-abdominal or retroperitoneal tumors	Massive fluid resuscitation or positive fluid balance	Sepsis
Laparoscopy with excessive insufflation pressures	Polytransfusion	Shock or hypotension
Liver dysfunction/cirrhosis with ascites		
Peritoneal dialysis		

contents, and (4) capillary leak and/or massive fluid/blood infusion.

Diminished abdominal wall compliance is typical in obese patients undergoing massive ventral hernia repair and in burned patients with skin retraction. Increased intraluminal contents occur in bowel obstruction and pseudo-obstruction. Increased intra-retroperitoneal contents typically happen when hemoperitoneum, retro-hemoperitoneum (typically caused by aorta aneurysm rupture), and intra- or extraperitoneal packing are present. Capillary leak and/or massive fluid/blood infusion can happen when shock gut is present or when more than 5 L of crystalloids or more than ten packed red blood cells units are administered in less than 24 h.

The complete list of risk factors for ACS developed by the WCACS 2013 [4] is reported in Table 49.2.

49.2.3 Diagnosis

To make the diagnosis of ACS, a high index of suspicion is necessary: in patients presenting one or more risk factors, IAP should be assessed at least every 8 h.

Clinical exam alone has a poor diagnostic power [5, 6], and the bladder pressure measurement is considered the diagnostic gold standard by many authors. The measurement should be performed with the patient in a supine position: after complete voiding of the bladder, 50–100 mL of saline solution should be instilled through the catheter to fill it partially. The goal is to have the bladder not collapsed or distended (and contracting) either. The column of liquid in the catheter should be connected with a transducer previously zeroed at the level of the pubic symphysis: the revealed pressure is a close approximation of the IAP.

49.2.4 Effects of IAH on Individual Systems

Respiratory system: Increasing IAP determines progressive diaphragm elevation; this results in decreased chest and lung compliance, reduced tidal volumes, and increased airway pressure. In these settings, the pulmonary function could be impaired, and the patient could suffer from hypercapnia (determining acidosis) and hypoxia and could develop respiratory failure.

Cardiovascular system: On one hand, blood return from inferior vena cava diminishes if IAP increases; on the other, elevated intrathoracic pressure and augmented intra-abdominal resistances increase the cardiac post-load; the result is a reduction in stroke volume. The heart compensates increasing its rate at first, but eventual dropping in cardiac output is likely. The concomitant hypercapnia and the subsequent acidosis could aggravate the cardiac failure by inhibiting the whole cardiovascular system.

In these settings, CVP and pulmonary artery wedge pressure are elevated and don't reflect the intravascular volume status anymore.

Renal system: IAH affects renal system by a mechanism which is not completely clear: probably multiple events occur resulting in glomerular filtration rate (GFR) reduction. IAH seems to reduce blood flow in renal veins; the resulting renal congestion seems to be responsible for renal edema, increased intracapsular pressure, and increased microcirculation resistance; these conditions justify the decrease in urinary output that could progress from oliguria to anuria and eventual kidney failure. Interestingly increased pressure on ureters doesn't seem to affect the renal function: ureteral stenting, while IAP is rising, doesn't impact on renal impairment.

Gastrointestinal system: As well as for kidneys, IAH results in gastrointestinal blood stream slowing down.

Low mesenteric flow could be demonstrated by gastric pH and tonometry variation. A recent study on laparoscopy showed that 15 mmHg intra-abdominal pressure is sufficient to reduce gut perfusion of 11–54%; that is why untreated ACS could easily lead to bowel ischemia and eventual death. In this setting, liver could suffer too: reduced portal flow from the gastrointestinal tract seems to be the main reason for liver impairment; no secure demonstration of this phenomenon has been made in animal or human model though.

CNS: IAH can cause a marked increase in intracranial pressure.

49.2.5 Treatment

Facing the rise of IAP could be challenging: the first goal of the treatment is to identify and correct any possible cause of ACS; this goal could be achieved by "non-operative management" and "surgical intervention.

Non-operative management pivots on four main elements [7]:

1. Decrease intraluminal gastrointestinal contents. The use of nasogastric/rectal tube or the use of gastro/colon prokinetic agents could be very helpful in these settings. Minimization of enteral nutrition and colonscopic decompression could be necessary as well.
2. Evacuation of the intra-abdominal space-occupying lesions. This goal could be achieved by percutaneous drainage of intra-abdominal collections.
3. Improve abdominal wall compliance. To achieve this goal, it is necessary to ensure adequate analgesia and sedation, to remove constrictive dressings and abdominal eschars, to avoid prone position (paying attention to maintain the head of bed >20°), and to consider neuromuscular blockade.
4. Optimize fluid management and perfusion. To achieve this goal, it is necessary to avoid excessive fluid administration aiming for negative balance, to use hypertonic fluids and colloids and to increase diuresis, to consider hemodialysis/ultrafiltration, and to maintain APP > 60 mmHg (with vasoactive medications when needed).

When non-operative management fails, IAH should be addressed by surgical intervention.

Common indications for surgery are:

1. IAP > 35 mmHg in repeated measurements
2. IAP 25–35 mmHg and progressive organ failure
3. Renal and pulmonary dysfunction
4. APP < 50 mmHg

Many techniques have been described to decompress the abdomen [8]; every technique has its own advantages and disadvantages as listed below. If IAH is following previous abdominal surgery, choosing the same incision is generally the best option.

1. Laparostomy: could be performed by midline incision (Fig. 49.2) (it is relatively safe, easy to perform, and nearly always effective; entero-atmospheric fistulas and high risk of persistent open abdomen are the main disadvantages) or by transverse subcostal incision (Fig. 49.3) (it is effective and has a higher rate of fascial closure; the major disadvantages are it is more time-consuming, and if fascial closure can't be achieved, complex reconstructions with loss of abdominal muscle function are nearly always unavoidable).

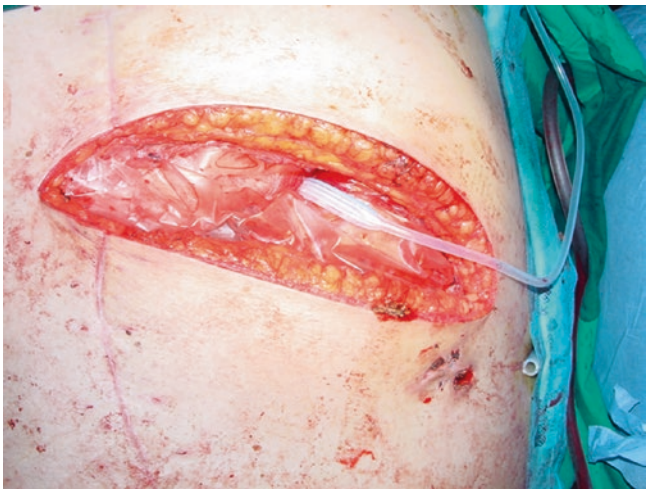

Fig. 49.2 Midline incision

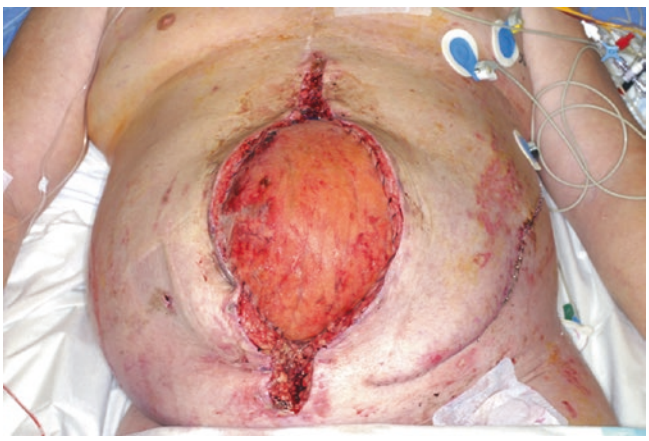

Fig. 49.3 Transverse subcostal incision

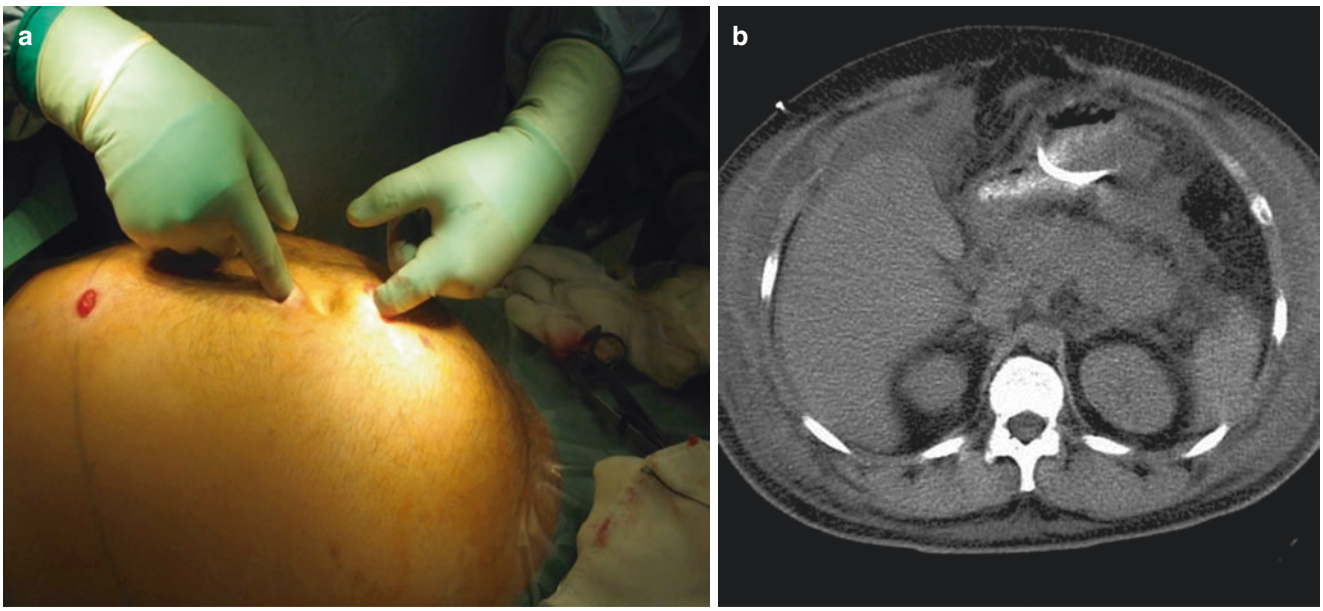

Fig. 49.4 Subcutaneous linea alba fasciotomy: performing the procedure (**a**), CT scan after the procedure (**b**)

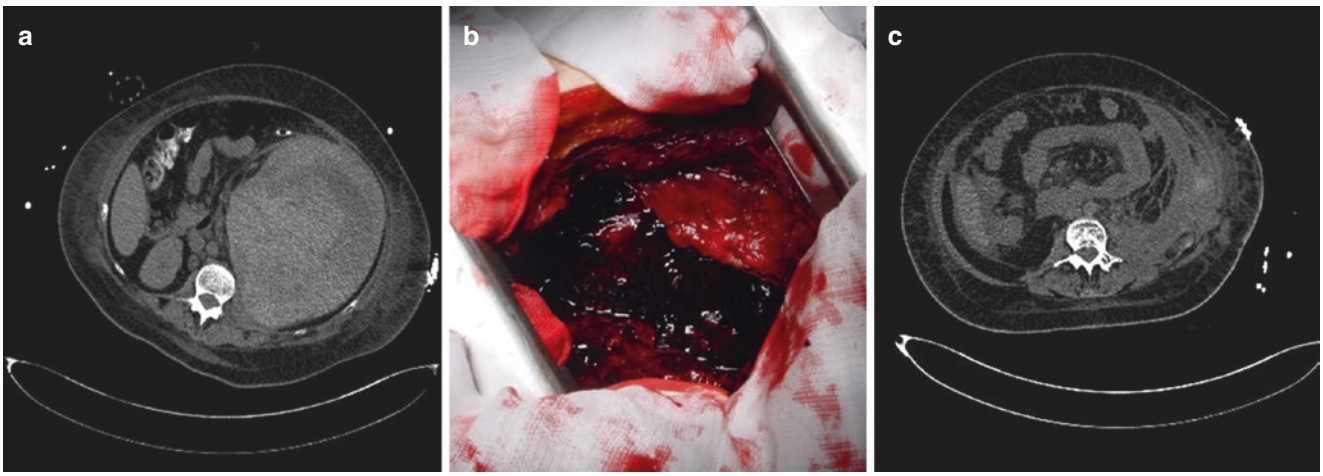

Fig. 49.5 Extraperitoneal posterior decompression: huge extraperitoneal hematoma at the CT scan (**a**); the surgical field (**b**); CT scan after decompression (**c**)

2. Subcutaneous linea alba fasciotomy (SLAF) (Fig. 49.4): through two or three short horizontal skin incisions, subcutaneous anterior abdominal fasciotomy at the linea alba is performed. The peritoneum is left intact. SLAF is effective in about 50–70%; it prevents open abdomen and its related complications, but it is always associated with a subsequent hernia. The cost-effectiveness (due to the lesser need of nursing care) is an advantage.
3. Extraperitoneal posterior decompression (Fig. 49.5): it is performed to evacuate a retroperitoneum mass (generally a hematoma) compressing the abdomen. It is effective and prevents open abdomen. The main disadvantage is it can be performed only if an extraperitoneal mass is causing IAH.

Independently from the chosen technical approach, the surgery could result in three physiological changes:

1. A sudden drop in systemic vascular resistance
2. A fall in intrathoracic pressure
3. A washout of toxic products (lactic acid and potassium)

These changes could lead to the potentially dangerous "abdominal decompression syndrome." This syndrome

is characterized by arrhythmias, myocardial depression, hypotension, and possibly barotrauma/volutrauma (due to the administration of a suddenly inadequate tidal volume).

To avoid this syndrome, a careful anesthesiologic conduct and effective communication between surgeon and anesthetist are mandatory during every phase of the decompressive operation.

49.3 Acute Compartment Syndrome of the Limbs

49.3.1 Definition

With the understanding of the pathophysiology underneath the Volkmann's syndrome, the definition of acute compartment syndrome (ACS) evolved, and the two classical signs (paralysis and a subsequent flexion contracture of the hand and forearm) have been replaced by the presence of a compartment pressure equal or bigger than the blood perfusion pressure.

49.3.2 Common Etiology and Risk Factors

The most common cause of ACS is trauma of the limbs. In a Scottish study on 164 patients presenting ACS, they found that 69% of them had fractures (being diaphyseal tibial fractures the most frequent (36%) followed by fractures of the distal radius (9.8%)) [9].

In the absence of fracture, older age, vascular injuries, and ischemia/reperfusion episodes are at higher risk for acute compartment syndrome development.

Elderly individuals found lying down on the ground with several hours of immobility should raise suspicion for compartment syndrome: the low blood flow determined by the prolonged contact with a hard surface could lead to swelling of a limb and trigger the pathological process. Similarly, vascular injuries and ischemia/reperfusion episodes trigger ACS in about 50% of the cases [10], and high suspicion is mandatory [11].

49.3.3 Diagnosis

The diagnosis of acute compartment syndrome of the limb is based on physical exam. The hallmark symptoms of the syndrome are the 6 P's: pain with passive stretch, pulselessness, pallor, poikilothermia, paresthesias, and paralysis.

Extreme pain (more than expected) seems to be the most reliable symptoms in early stages of ACS and should raise suspicion. Comparison with a contralateral limb is important too: extremities with impending or active compartment syndrome are often swollen and shiny compared to the uninjured extremity.

The instrumental diagnosis of acute compartment syndrome of the limb is not mandatory but could be achieved by needle manometer (using an 18-g straight needle, side-port needle, slit catheter, or wick catheter). The measurement should be taken with the patient in a supine position; the measured extremity should be at the heart level; the needle should be placed perpendicular to muscle belly, near to the fracture site or the area of maximum tension. Needle measurement is contraindicated when underlying neurovascular structures are at risk; the patient suffers from coagulopathy or local diffuse cellulitis.

In a paper from 1979 [12], Hargens showed that intracompartmental pressures of 120 mmHg could lead to complete nerve blockage in less than 2 h; pressures around 30–40 mmHg could determine partial nerve blockage in 6–8 h; pressures lower than 20 mmHg didn't determine nerve conduction impairment in any conditions. On the basis of these observations, the authors suggested that 30 mmHg should be considered the critical compartment pressure to start the treatment.

49.3.4 Treatment

Treatment for acute compartment syndrome starts with removal of any cast, splint, or bandage from the affected limb. Then fasciotomies should be performed to decompress any compartment affected by the syndrome. Table 49.3 shows the compartments of the upper limb and Table 49.4 the compartments of the lower limb.

Table 49.3 Compartments of the upper limb

Region	Compartments	
Arm	Anterior	
	Posterior	
	Deltoid[a]	
Forearm	Volar	Superficial deep
	Dorsal	Mobile wad
	Hand	Extensor anconeus
Hypothenar	Thenar	
	Adductor pollicis	
	Dorsal interossei	
	Volar interossei	
	Finger (x5)	

[a]Deltoid doesn't lie in a real compartment, but its fascia is thick and may need decompression

Table 49.4 Compartments of the lower limb

Region	Compartments	
Thight	Anterior	
	Medial	
	Posterior	
Leg	Anterior	
	Lateral	
	Poterior	Superficial Deep
Foot	Medial	
	Lateral	
	Interosseus (×4)	
	Central	Superficial Central Deep

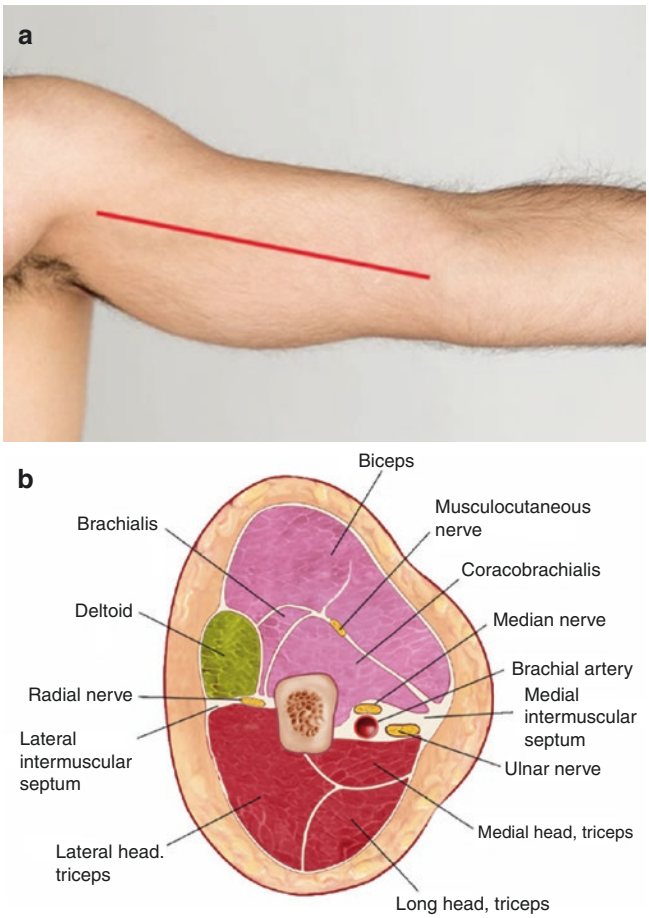

Fig. 49.6 Arm decompression. The anterior and posterior compartments could be decompressed by a single median incision (**a**). Compartments of the arm (**b**): in red the posterior compartment, in purple the anterior compartment, in green the deltoid compartment. With permission of Springer Nature; from Patel K., Major N. (2009) Compartmental Anatomy. In: Davies A., Sundaram M., James S. (eds) Imaging of Bone Tumors and Tumor-Like Lesions. Medical Radiology. Springer, Berlin, Heidelberg

The anterior and posterior compartments of the arm can be decompressed by a single median incision (Fig. 49.6). Once the fascia and the lateral intermuscular septum are identified, two longitudinal incisions in the fascia overlying the anterior and posterior compartments are made.

The forearm can be decompressed by two incisions as shown in Fig. 49.7. On the volar aspect of the forearm, the incision is curvilinear, starts at the antecubital fossa, and goes down toward the mid-palm. If necessary, the incision is carried further distally to release the carpal tunnel as well. The second incision decompresses the dorsal compartment: it starts from the lateral epicondyle, goes straight down at the center of the forearm, and ends at the center of the wrist.

Hand compartment decompression lines are shown in Fig. 49.8.

Decompressing the anterior and posterior compartments by a single incision is sufficient most of the time since the medial compartment is rarely affected by the ACS. The incision is shown in Fig. 49.9. Once the fascia lata is opened, the anterior compartment is decompressed. Dissection and elevation of the vastus lateralis give access to the intermuscular septum. The incision of this septum decompresses the posterior compartment.

Leg decompression can be obtained by two different techniques. The first is based on a single incision: it is made directly over the fibula; the anterior and lateral compartments are accessed elevating a skin flap anteriorly; and the superficial and deep posterior compartments are accessed by elevating a posterior flap.

The second technique is based on two incisions (Fig. 49.10). The medial incision is 3 cm posterior to the tibia and allows access to the superficial and deep posterior compartments. The lateral incision is centered between the tibial crest and fibular shaft and allows access to the anterior and lateral compartments. Care should be taken while making this access to preserve the superficial peroneal nerve.

The decompression of the foot is controversial since the burden of long-term procedure complications could overcome the attended benefit from the surgery. The procedure is performed through two dorsal incisions and a single medial incision. The dorsal incisions are made over the second and fourth metatarsals allowing for decompression of the interosseous, central, medial, and lateral compartments. The third incision is made on the medial aspect of the foot, inferior to the first metatarsal allowing better decompression of the central compartment.

A delay in treating an acute compartment syndrome could be dramatic and lead to permanent disability, amputation, or even death [3]. That is why prompt recognition and treatment (possibly within 6 h) of the ACS should be considered a priority.

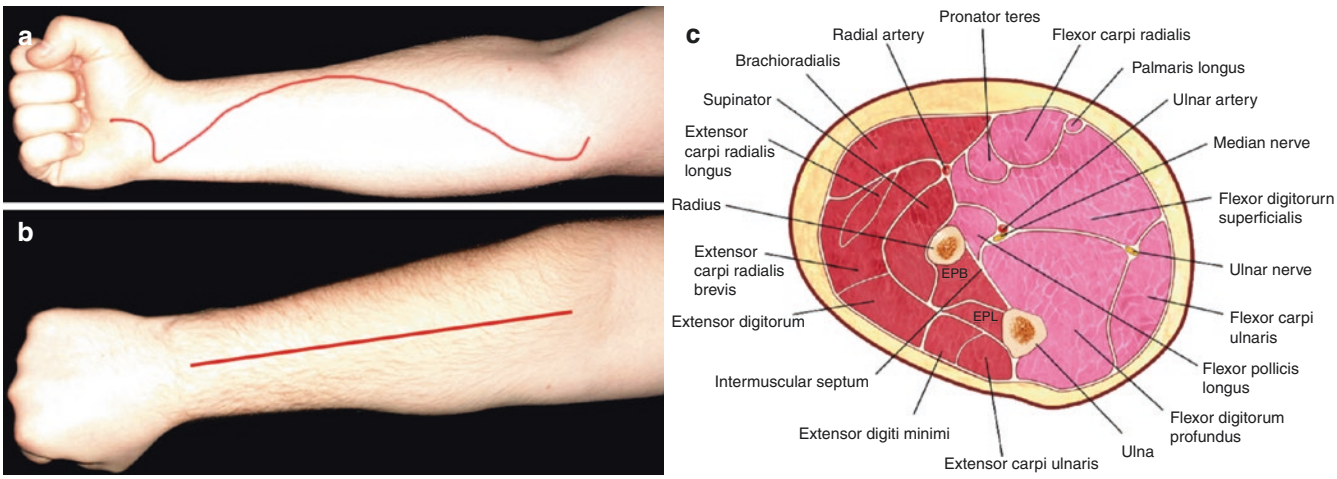

Fig. 49.7 Forearm decompression: volar incision (**a**), dorsal incision (**b**), compartments (**c**): in purple the volar compartment, in red the dorsal compartment. With permission of Springer Nature; from Patel K., Major N. (2009) Compartmental Anatomy. In: Davies A., Sundaram M., James S. (eds) Imaging of Bone Tumors and Tumor-Like Lesions. Medical Radiology. Springer, Berlin, Heidelberg

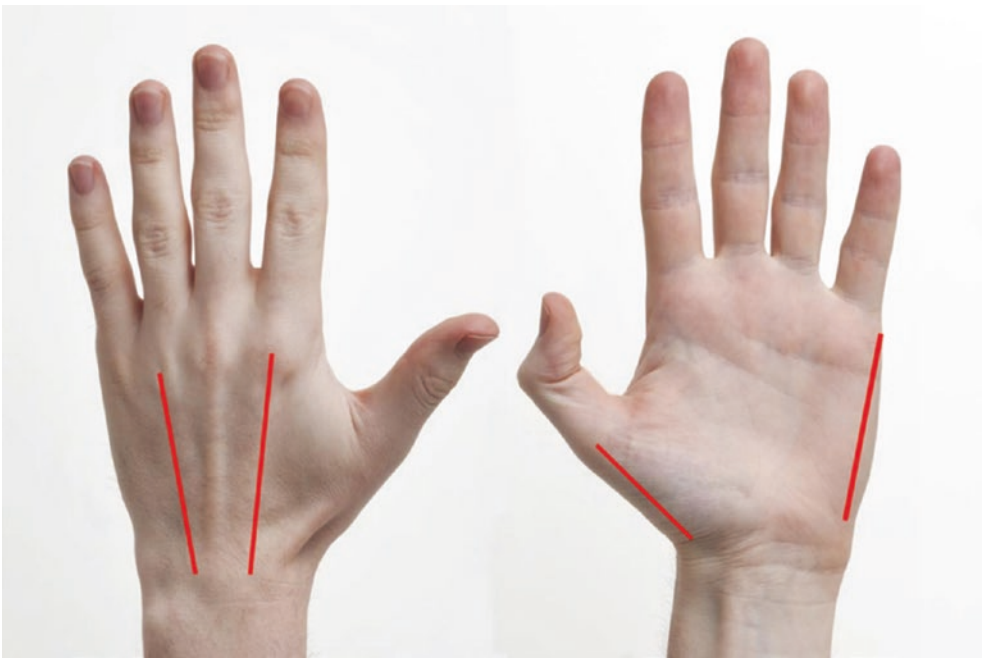

Fig. 49.8 Incision lines to decompress the hand compartments

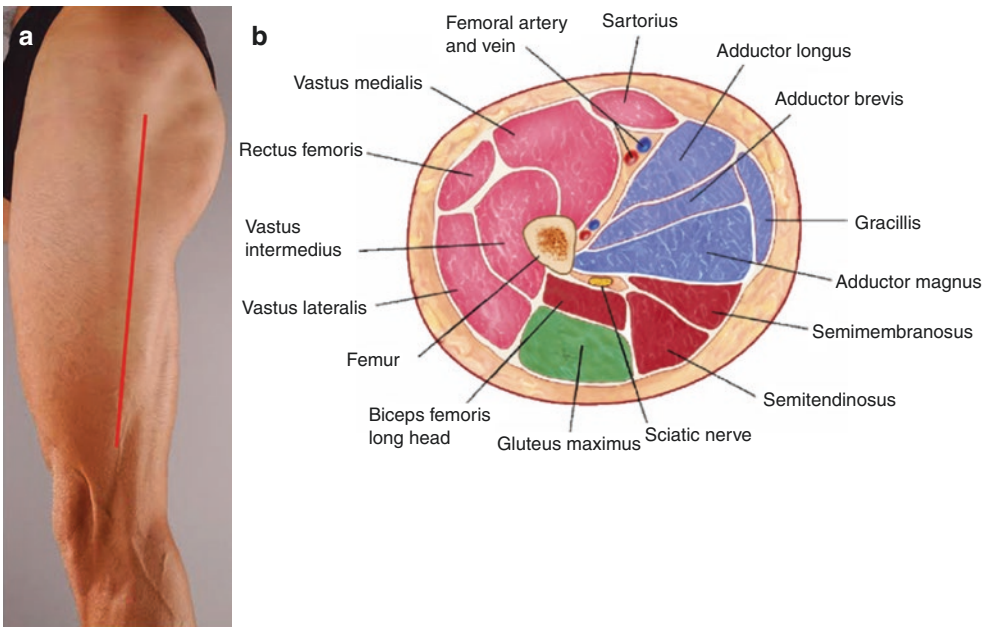

Fig. 49.9 Tight incision line (**a**); tight compartments (**b**): in purple the anterior compartment, in blue the medial compartment, in red the posterior compartment. With permission of Springer Nature; from Patel K., Major N. (2009) Compartmental Anatomy. In: Davies A., Sundaram M., James S. (eds) Imaging of Bone Tumors and Tumor-Like Lesions. Medical Radiology. Springer, Berlin, Heidelberg

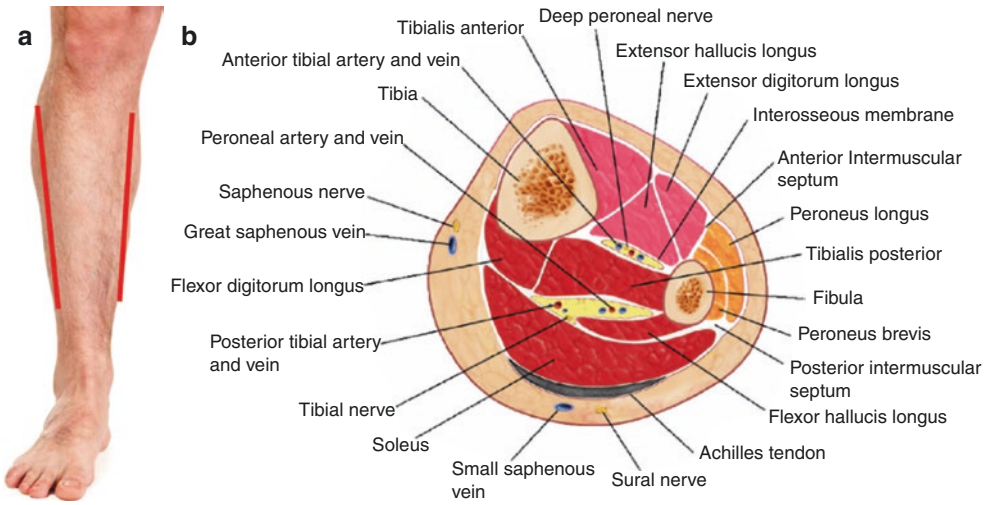

Fig. 49.10 Leg incision lines (**a**); leg compartments (**b**): in purple the anterior compartment, in orange the lateral compartment, in red the posterior compartment. With permission of Springer Nature; from Patel K., Major N. (2009) Compartmental Anatomy. In: Davies A., Sundaram M., James S. (eds) Imaging of Bone Tumors and Tumor-Like Lesions. Medical Radiology. Springer, Berlin, Heidelberg

Case Scenario

We present the case of a 56-year-old male undergoing liver transplant for HCC on HCV cirrhosis. The hepatectomy and implantation were uneventful; vitals were stable during the whole procedure, and blood loss was minimal (300 mL). After the reperfusion, the graft swelled up making the abdomen closure tricky because of a slight tension of the wound. Within 6 h after the surgery, the abdomen became distended, and the respiratory and kidney functions got impaired. Systolic tension was stable at 100 mmHg while diastolic was 85 mmHg. Lactates were stably 4.5 mmol/L. IAP (measured by transurethral probe) was increasing: when it reached 26 mmHg, we performed a CT scan showing intra-abdominal clots with no sign of active bleeding. The patient was taken to the operatory room: after clot removal (Fig. 49.11) and accurate hemostasis, a Bogota bag was placed and an open treatment performed. After 48 h, the liver, kidney, and respiratory functions were normal, and the wound could be closed without any other complications.

Fig. 49.11 Case scenario: clots removed during open treatment for ACS

Questions:

1. What are the clinical signs that could have raised the suspicion of ACS?
   A. The distended abdomen
   B. Respiratory and kidney function impairment
   C. Low blood tension and reduced lactate clearance
   D. All of the above
2. The impaired kidney function is caused by:
   A. Decreased blood flow in renal veins and kidney congestion
   B. Compression on the ureters
   C. Increased blood catabolite concentration
   D. All of the above
3. The impaired respiratory function is caused by:
   A. Diaphragm elevation
   B. Reduced chest and lung capacity
   C. Increased airway pressure
   D. All of the above
4. In the present case, the most important element to confirm the ACS was:
   A. The clinical sign
   B. The increased bladder pressure
   C. The CT scan
   D. The lactate level
5. What was the main goal of the surgery in the present case?
   A. Check the anastomosis
   B. Reduce the IAP
   C. Avoid early incisional hernia
   D. All of the above

Please see Chap. 58 for the correct answer.

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